U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 11 December 2003

Case No.: 2001-BLA 537

In the Matter of:

NELDA J. MCBRAYER Survivor of ROY MCBRAYER,
Claimant

v.

ISLAND CREEK COAL COMPANY,
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-in-Interest

Appearances:

Joseph E. Wolfe, Esquire
For the Claimant

Mary Rich Maloy, Esquire
For the Employer

Before: Robert J. Lesnick
Administrative Law Judge

DECISION AND ORDER – DENYING BENEFITS

This case arises from a claim for benefits under the "Black Lung Benefits Act," Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended, 30 U.S.C. §901 *et seq.* (hereinafter referred to as "the Act"), and applicable federal regulations, mainly 20 C.F.R. Parts 718 and 725 ("Regulations"). [1]

[1] The Secretary of Labor adopted amendments to the "Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969" as set forth in Federal Register/Vol. 65, No. 245 Wednesday, December 20, 2000. The amended Part 718 Regulations became effective on January 19, 2001, and were to apply to both pending and newly filed cases. The new Part 725 Regulations also became effective on January 19, 2001. Some of the new procedural aspects of the Part 725 Regulations, however, were to apply only to claims filed on or after January 19,

Benefits under the Act are awarded to persons who are totally disabled, within the meaning of the Act, due to pneumoconiosis, or to the survivors of persons whose death was caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as black lung.[2]

A formal hearing was scheduled for April 15, 2003, in Charleston, West Virginia. On March 28, 2003, counsel for Claimant submitted a Motion to Have Hearing on the Record. Counsel for Employer had no objection, and on April 3, 2003, I issued an Order Granting Decision on the Record. [3] On June 9, 2003, Claimant filed her closing brief and Employer filed its closing brief.

ISSUES

The contested issues are:

1. Length of the miner's coal mine employment;
2. Whether the miner had pneumoconiosis;
3. Whether the pneumoconiosis arose out of the miner's coal mine employment; and
4. Whether the miner's death was due to pneumoconiosis.

2001, not to pending cases. The Amendments to the Part 718 and 725 Regulations were challenged in a lawsuit filed in the United States District Court for the District of Columbia in *National Mining Association v. Chao*, No. 1:00CV03086 (EGS). On February 9, 2001, the District Court issued a Preliminary Injunction Order that enjoined the application of the Amendments "except where the adjudicator, after briefing by the parties to the pending claim, determines that the regulations at issue in the instant lawsuit will not effect the outcome of the case." On August 9, 2001, the United States District Court for the District of Columbia issued a decision granting the U.S. Department of Labor's motion for summary judgment in *National Mining Association v. Chao*, dissolved the preliminary injunction, and upheld the validity of the amended regulations. The case was subsequently appealed and on June 14, 2002, the United States Court of Appeals for the District of Columbia Circuit issued a decision that affirmed in part, reversed in part and remanded the case back to the District Court for further instruction. The Court of Appeals upheld the validity, for the most part, of the challenged amendments except that the Court found the following sections to be impermissibly retroactive: §§718.204(a), 725.701, 725.101(a)(31), 725.204, 725.212(b), 725.213(c), 725.214(d), 725.219(c) and (d). The Court also found §725.101(a)(6) to be invalid.

[2] The following abbreviations have been used in this opinion: DX = Director's exhibit, EX = Employer's exhibit, CX = Claimant's exhibit, TR = Transcript of the hearing, BCR = Board-certified radiologist, BCI = Board-certified internist, and B = B reader.

[3] Director's exhibits 1 through 43, and Employer's exhibits 1 through 17 are hereby admitted into evidence without objection. Employer's pre-hearing report, dated April 18, 2003, is marked ALJ-1.

(DX 42; ALJ-1)

FINDINGS OF FACT

Procedural History and Factual Background[4]

The miner, Roy L. McBrayer, filed his claim for Black Lung benefits on October 10, 1989. (DX 41-1). The claim was denied by the District Director on March 29, 1990. (DX 41-16). The miner requested a formal hearing and on February 26, 1992, a hearing was held before Administrative Law Judge Eric Feirtag in Charleston, West Virginia. At the hearing, the parties stipulated to 40 years of coal mine employment, that the miner suffered from a totally disabling respiratory impairment, and that he had one dependent, his wife, for purposes of augmentation of benefits. (DX 41-64). On May 28, 1992, Judge Feirtag issued a Decision and Order Denying Benefits. In his decision, Judge Feirtag found the miner had established the existence of pneumoconiosis based on the x-ray evidence (§718.202(a)(1)) but that the miner failed to establish his totally disabling respiratory impairment was due to pneumoconiosis. (DX 41-65). The miner appealed and Employer cross-appealed the decision to the Benefits Review Board ("BRB"). The miner challenged the Judge's findings regarding causation of total disability while Employer challenged the Judge's findings regarding the existence of pneumoconiosis pursuant to §718.202(a)(1). On October 4, 1993, the BRB issued a Decision and Order affirming Judge Feirtag's findings. (DX 41- 66). No further action was taken on this claim.

The miner died on May 16, 1997. On February 9, 1999, Nelda McBrayer ("Claimant") filed a claim for survivor's benefits. (DX 1). The miner and Claimant were married on June 22, 1968. (DX 8). The miner was divorced from his first wife, Mary Susan McBrayer, on January 12, 1968. (DX 10). On July 7, 1999, a Claims Examiner denied Claimant's claim for benefits. (DX 18). After the submission of additional evidence, on December 21, 2000, the District Director awarded benefits to Claimant. (DX 32). Employer disagreed with the determination and requested a formal hearing. (DX 36). The case was transferred to the Office of Administrative Law Judges on March 7, 2001. (DX 43). After several continuances, a hearing was finally set for April 15, 2003, in Charleston, West Virginia. However, at the request of Claimant, the hearing was cancelled and a decision will be rendered based on the record.

Deposition of Claimant

The deposition of Claimant was taken on July 2, 1998, and appears at DX 24. Claimant testified that she was married to the miner for 28 years. At the time of his death, the couple had no dependent children. She stated the miner was hospitalized 40 days prior to his death, but died two hours after they sent him home. She stated the miner's treating physician was Dr. Stanley. She noted that Dr. Stanley treated the miner for his high blood pressure, breathing, and cough.

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[4] Given the filing date of this claim, subsequent to the effective date of the permanent criteria of Part 718, (*i.e.* March 31, 1980), the Regulations set forth at 20 C.F.R. Part 718 will govern its adjudication. Because the miner's last exposure to coal mine dust occurred in West Virginia this claim arises within the territorial jurisdiction of the United States Court of Appeals for the Fourth Circuit. *See Broyles v. Director, OWCP*, 143 F.3d 1348, 21 BLR 2-369 (10th Cir. 1998).

She added that Dr. Stanley treated the miner with an inhaler. Claimant noted the miner had pneumonia on about four occasions. She stated the miner worked 41 years and nine months in the coal mines. The last ten years of his coal mine employment was underground. Claimant stated the miner stopped smoking in 1993, and that he had smoked two packs of filtered cigarettes per day for more than 20 years. Just before his death, the miner was on various medications prescribed by Dr. Stanley. She added the miner was on supplemental oxygen at home for three years prior to death. She noted that he was in a coma for three days before his death. Claimant stated that by 1993, the cancer was 95% cured and that it had not come back.

Medical Evidence

A. Chest X-rays[5]

X-ray	Exhibit	Physician/Qualifications	Interpretation
3-11-83	DX 24	Goerlich	mild pulmonary em and interstitial pulmonary fibrosis
2-20-84	DX 28	Wiot/BCR, B	no CWP; bu; em
2-20-84	DX 26	Spitz/BCR, B	Negative
2-20-84	DX 34	Shipley/BCR, B	no CWP; em
2-20-84	EX 9	Fino/B	0/0
1-3-85	DX 41-40	Stahly	no active disease; possible COPD
10-30-86	DX 41-40	Stahly	right ribs: normal
9-21-89	DX 41-11	Breyfogle	probable nipple shadow; nodular

[5] A=A-reader; B=B-reader; BCR=Board-Certified Radiologist; R=Radiologist; BCP=Board-Certified Pulmonologist; BCI=Board-Certified Internal Medicine; BCCC=Board-Certified Critical Care. Readers who are board-certified radiologists and/ or B-readers are classified as the most qualified. B-readers need not be radiologists. The existence of pneumoconiosis may be established by chest x-rays classified as category 1, 2, 3, A, B, or C according to ILO-U/C International Classification of Radiographs. A chest x-ray classified as category 0, including subcategories 0/-, 0/0, 0/1, does not constitute evidence of pneumoconiosis. 20 C.F.R. § 718.102(b).

			density not absolutely excluded; patchy atelectasis, left base
9-21-89	DX 41-39	Abramowitz/BCR, B	0/1, s/t, 4 zones
9-21-89	DX 41-39	Duncan/BCR, B	0/1, s/t, 4 zones
9-21-89	DX 41-39	Binns/BCR, B	no CWP
9-22-89	DX 41-11	Breyfogel	asthmatic bronchitis
9-22-89	DX 41-39	Abramowitz/BCR, B	0/1, s/t, 4 zones
9-22-89	DX 41-39	Duncan/BCR, B	0/1, s/t, 4 zones; em
9-22-89	DX 41-39	Binns/BCR, B	Negative
9-27-89	DX 41-40	Shah/BCR, B	pneumonic infiltrate with associated atelectasis RML
9-29-89	DX 41-11 DX 41-40	Shah/BCR, B	partial resolution of pneumonic infiltrate; another ill-defined increased density LLL
10-3-89	DX 41-11 DX 41-40	Shah/BCR, B	resolution of pneumonic infiltrates in RML and LLL
12-7-89	DX 41-15	Shah/BCR, B	0/1, s/s, 4 zones
12-7-89	DX 41-14	Gaziano/B	Negative
12-7-89	DX 41-39	Hayes/BCR, B	Negative
12-7-89	DX 41-39	Abramowitz/BCR, B	Negative
12-7-89	DX 41-39	Duncan/BCR, B	no CWP; em
12-7-89	DX 41-39	Binns/BCR, B	Negative
12-7-89	DX 28	Wiot/BCR, B	no CWP; od

12-7-89	DX 26	Shipley/BCR, B	Negative
12-7-89	DX 26	Spitz/BCR, B	Negative
12-7-89	EX 9	Fino/B	0/0
8-1-90	DX 41-34	Zaldivar/B	no CWP
8-1-90	DX 41-36	Fino/B	no CWP
8-1-90	DX 41-37	Renn/B	Negative
8-1-90	DX 41-43	Wheeler/BCR, B	no CWP
8-1-90	DX 41-43	Scott/BCR, B	no CWP
2-12-91	DX 24 DX 41-24	Speiden/BCR, B	1/1, p/q, 6 zones
2-12-91	DX 24 DX 41-26	Ahmed/BCR, B	2/1, p/q, em
2-12-91	DX 24 DX 41-27	Pathak/BCR, B	2/2, p/q, em
2-12-91	DX 24 DX 41-28	Cappiello/BCR, B	2/1, p/q, em
2-12-91	DX 24 DX 41-29	Aycoth/BCR, B	2/2, q/p, em
2-12-91	DX 41-11	Zaldivar/B	Negative
2-12-91	DX 41-42	Wheeler/BCR, B	no CWP
2-12-91	DX 41-42	Scott/BCR, B	no CWP
2-12-91	DX 41-42	Gayler	no CWP
1-29-92	DX 41-46	Zaldivar/B	Negative

5-22-92	DX 28	Wiot/BCR, B	no CWP; bu; od
5-22-92	DX 26	Spitz/BCR, B	Negative
5-22-92	DX 34	Shipley/BCR, B	no CWP; em
5-22-92	EX 9	Fino/B	0/0
7-27-93	DX 29	Zaldivar/B	no CWP; bu; comment
7-27-93	DX 27	Wheeler/BCR, B	no CWP; od; comment
7-27-93	DX 27	Scott/BCR, B	no CWP; em; comment
3-14-94	DX 37	Shah/BCR, B	mild congestive HF; rounded nodular density LLL; chronic COPD
3-14-94 CT	DX 37	Shah/BCR, B	small cyst upper pole left kidney
3-14-94 CT	EX 3	Wiot/BCR, B	no CWP; mild em
3-14-94 CT	EX 1	Spitz/BCR, B	no nodules or eggshell calcifications mass on left
3-14-94 CT	EX 6	Perme/BCR, B	no CWP; bilateral lung infiltrates, mild lymphadenopathy, possible organizing pneumonia; RLL liver lesion
3-14-94 CT	EX 9	Fino/B	Negative
3-20-94	DX 37	Shah/BCR, B	minimal improvement of congestive HF since 3-14-94
3-25-94	DX 37	White	no change over last 5 days compatible w/ chronic pulmonary disease

6-17-94	DX 29	Zaldivar/B	no CWP; comments
6-17-94	DX 27	Wheeler/BCR, B	no CWP; od; comment
6-17-94	DX 27	Scott/BCR, B	no CWP; od; em; comment
6-17-94	EX 9	Fino/B	0/0
6-17-94	EX 14	Wiot/BCR, B	no CWP; bu; em; od; comment
6-17-94	EX 16	Spitz/BCR, B	no CWP; em; comment
6-17-94	EX 16	Shipley/BCR, B	no CWP; comments
9-1-95	DX 28	Wiot/BCR, B	no CWP; bu; od; comment
9-1-95	DX 26	Spitz/BCR, B	no CWP; comments
9-1-95	DX 34	Shipley/BCR, B	no CWP; em
9-1-95	EX 9	Fino/B	0/0
9-1-95	EX 13	Wheeler/BCR, B	no CWP; od; em(?) comments
9-1-95	EX 13	Scott/BCR, B	no CWP; od; em(?) comments
6-7-96	DX 29	Zaldivar/B	u/r
6-7-96	DX 27	Wheeler/BCR, B	no CWP; od; co; kl comments
6-7-96	DX 27	Scott/BCR, B	no CWP; co ;comment
6-7-96	EX 9	Fino/B	0/0
6-9-96	DX 29	Zaldivar/B	no CWP; ef; comment
6-9-96	DX 27	Wheeler/BCR, B	no CWP; od; kl(?) comments

6-9-96	DX 27	Scott/BCR, B	no CWP; od; ef; em comments
6-9-96	EX 14	Wiot/BCR, B	no CWP; bu; em; od comments
6-9-96	EX 16	Spitz/BCR, B	no CWP; em; comment
6-9-96	EX 16	Shipley/BCR, B	no CWP; comments
7-24-96	DX 29	Zaldivar/B	no CWP; bu; comment
7-24-96	DX 27	Wheeler/BCR, B	no CWP; od; kl(?) comments
7-24-96	DX 27	Scott/BCR, B	no CWP; od; em; comments
7-24-96	EX 9	Fino/B	0/0
7-24-96	EX 14	Wiot/BCR, B	no CWP; bu; em; od comments
7-24-96	EX 16	Spitz/BCR, B	no CWP; em; comment
7-24-96	EX 16	Shipley/BCR, B	no CWP; comments
8-1-96	DX 29	Zaldivar/B	no CWP; comments
8-1-96	DX 27	Wheeler/BCR, B	no CWP; od; ef(?); em comments
8-1-96	DX 27	Scott/BCR, B	no CWP; od; ef(?); em comments
8-1-96	EX 9	Fino/B	0/0
8-1-96	EX 14	Wiot/BCR, B	no CWP; bu; em; od; comments
8-1-96	EX 16	Spitz/BCR, B	no CWP; em; comment
8-1-96	EX 16	Shipley/BCR, B	no CWP; comments

1-6-97 CT	DX 39	Zarabi	centrilobular em;mass LLL possible malignancy; linear densities likely due to pulmonary fibrosis
1-6-97 CT	EX 3	Wiot/BCR, B	no CWP; em; patchy areas of infiltrate; nondescript mediastinal nodes
1-6-97 CT	EX 1	Spitz/BCR, B	no pulmonary nodules or eggshell calcifications; bilateral infiltrate and mediastinal nodules
1-6-97 CT	EX 6	Perme/BCR, B	no CWP; bilateral infiltrate most likely to infectious etiology; LLL liver lesion
1-6-97 CT	EX 9	Fino/B	Negative
1-6-97 CT	EX 13	Wheeler/BCR, B	no CWP; coarse infiltrates or fibrosis LLL involving pleura compatible with inflammatory disease
1-6-97 CT	EX 13	Scott/BCR, B	no CWP; em; infiltrate or scarring LLL; minimal infiltrate RLL; few enlarged mediastinal nodes
1-6-97 CT	EX 15	Fishman/BCR, B	Emphysema; possible aspiration pneumonia LLL; no CWP
1-17-97	DX 29	Zaldivar/B	no CWP; bu; comment
1-17-97	DX 27	Wheeler/BCR, B	no CWP; od; comment
1-17-97	DX 27	Scott/BCR, B	no CWP; od; em; comments
1-17-97	EX 9	Fino/B	0/0
1-17-97	EX 14	Wiot/BCR, B	no CWP; bu; em; od;

			comments
1-17-97	EX 16	Spitz/BCR, B	no CWP; em; comment
1-17-97	EX 16	Shipley/BCR, B	no CWP; comments

B. Pulmonary Function Studies

Exhibit No.	Date	Age	Height[6]	FEV1	MVV	FVC	Qualify
DX 24	3-11-83	55	68”	1.67	--	2.56	No
DX 24	2-20-84	57	66”	2.14	90	3.48	No
DX 24	12-7-89	62	68”	1.51	62	2.54	Yes
DX 41-34	8-1-90	63	65.25”	2.70 1.46*	46 70*	3.81 2.80*	No No
DX 24	1-14-92	64	66”	1.59	58	2.69	Yes
DX 41-46	1-29-92	64	65.25”	2.81	52	1.45	No
DX 24	5-22-92	65	66”	1.30	49	2.44	Yes
DX 24	10-27-92	65	66”	1.50	46	2.63	Yes
DX 24	2-24-93	65	66”	1.40	58	2.73	Yes

*Post-bronchodilator

C. Arterial Blood Gas Studies

Exhibit No.	Date	pO_2	pCO_2	Qualify
DX 41-35	5-11-83	76.9	40	No
DX 24	9-21-89	60 49	47 47	Yes Yes

[6] The average height for the miner is 66.28 inches. This height is used to determine whether the values are qualifying for impairment under the Regulations.

DX 24	9-22-89	49	48	Yes
DX 24	12-7-89	65 74*	44 43*	No No
DX 41	8-1-90	57	45	Yes
DX 24	1-14-92	57	44	Yes
DX 41-46	1-29-92	59	43	Yes
DX 37	5-1-94	65.5	42.3	No
DX 38	4-13-96	91.1	47.3	No
DX 39	12-11-96	39	37	Yes
DX 12	1-4-97	57	39	Yes
DX 12	1-4-97	40.7	38	Yes
DX 39	3-14-97	40	46	Yes

*Post-exercise

D. Medical Reports

Drs. J.M. Previll and J.L. Leef

The medical report of Drs. Previll and Leef is dated February 20, 1984, and appears at DX 41-35. They noted an occupational history of thirteen (13) years of underground coal mine employment. The miner had a smoking history of eight (8) pack years, having quit three years prior to the examination. Physical examination revealed an increase in AP diameter with normal breath sounds. Pulmonary function studies were normal. Drs. Previll and Leef concluded there was no evidence of occupational pneumoconiosis.

Dr. Susan Wantz

The medical report of Dr. Wantz is dated December 7, 1989, and appears at DX 41-12. Dr. Wantz examined the miner at the request of the Department of Labor. She reviewed the miner's occupational history and noted a family history positive for high blood pressure, heart disease, asthma, allergies, and stroke. The miner reported a medical history of pneumonia, attacks of wheezing, arthritis, and high blood pressure. She noted a smoking history of one pack per day from 1969 and continuing. His chief complaints were sputum production, wheezing, cough, chest pain, orthopnea, and paroxysmal nocturnal dyspnea. Physical examination was unremarkable. A chest x-ray was positive for pneumoconiosis and congestive heart failure. Dr.

Wantz diagnosed the miner as having: (1) severe chronic obstructive pulmonary disease ("COPD") based on history, physical examination, and pulmonary function studies, (2) congestive heart failure based on chest x-ray and history, and (3) possible coronary artery disease by history. Dr. Wantz attributed the COPD to coal mining and smoking. Dr. Wantz concluded the miner was not able to exert himself secondary to shortness of breath from severe COPD. Dr. Wantz opined the severe COPD was the major cause of impairment and that the congestive heart failure also contributed.

Dr. George Zaldivar

The medical report of Dr. Zaldivar is dated September 5, 1990, and appears at DX 41-34. Dr. Zaldivar is Board-Certified in Internal Medicine and Pulmonary Disease, and is a B-reader of chest x-rays. Dr. Zaldivar examined the miner on August 1, 1990. Dr. Zaldivar reviewed the occupational history of the miner and noted a smoking history of one (1) pack of cigarettes per day for twenty-seven (27) years. He also reviewed the hospital records from Dunlap Hospital, the medical report of Drs. Previll and Leef, and the medical report of Dr. Wantz. He reviewed various blood gas studies, vent studies, and various chest x-ray interpretations. Dr. Zaldivar's findings included: (1) high carboxyhemoglobin of a current smoker, (2) moderate reversible airways obstruction, (3) moderate resting hypoxemia, and (4) no radiographic evidence of pneumoconiosis. Dr. Zaldivar opined the miner had asthma which was "absolutely unrelated to coal mine work." He further stated the miner did not have coal workers' pneumoconiosis "neither radiographically, nor by history and physical examination." He noted that the miner's pulmonary impairment was variable depending on the status of the asthma. At the time of his examination, the asthma gave him a moderate impairment which would have prevented him from performing his last coal mine employment as a hand loader.

The second medical report of Dr. Zaldivar is dated January 29, 1992, and appears at DX 41-46. Dr. Zaldivar examined the miner on January 29, 1992. The miner's chief complaint was left leg pain and shortness of breath. The miner reported being short of breath for 15-20 years and noted that his breathing was worse in hot weather. He also had a cough productive of yellow sputum. Dr. Zaldivar noted a smoking history of 1½ packs per day from the age of 32, and an occupational history of forty-one (41) years of coal mine employment. Physical examination of the lungs was unremarkable after the inhalation of a bronchodilator. Dr. Zaldivar also reviewed additional medical evidence provided by Employer. Dr. Zaldivar concluded there was no radiographic evidence of pneumoconiosis. In addition, he noted the miner had severe resting hypoxemia, moderate irreversible airway obstruction, air trapping by lung volumes, and moderate diffusion impairment. Dr. Zaldivar opined that what began as asthma had now deteriorated into emphysema from his smoking habit. He added that the miner still smoked and continued to damage his lungs. He concluded that none of the pulmonary impairment was in any way related to the miner's occupation. He opined the miner was totally disabled from performing anything beyond strictly sedentary work.

The deposition of Dr. Zaldivar was taken on February 18, 1992, and appears at DX 41-63. Dr. Zaldivar testified that in order to diagnose "medical" coal workers' pneumoconiosis, one had to have a positive chest x-ray. In order to diagnose "black lung disease," one would have to consider "the history given by the individual, the breathing test changes, and Mr. McBrayer

doesn't meet either the arbitrary definitions of the black lung disease, nor does he meet the coal workers pneumoconiosis disease." (DX 41-63, pp. 76-77). The remainder of his testimony was basically a reiteration of his findings contained within his reports.

Dr. D.L. Rasmussen

The medical report of Dr. Rasmussen is dated April 10, 1991, and appears at DX 24. Dr. Rasmussen evaluated the miner on February 12, 1991. He reviewed the occupational history and noted about forty-one (41) years of coal mine employment. He noted that the miner's last employment was in September 1988. The miner complained of shortness of breath for about ten years, morning productive cough, wheezing, two-pillow orthopnea, occasional paroxysmal nocturnal dyspnea, and occasional chest pain. Physical examination revealed markedly reduced breath sounds, a prolonged expiratory phase, and expiratory wheezing with forced expiration. Dr. Speiden, who is a Board-Certified Radiologist and B-reader, read the chest x-ray as positive for pneumoconiosis (1/1, p/q, 6 zones). Dr. Rasmussen concluded the miner had a significant history of occupational dust exposure and x-ray changes consistent with pneumoconiosis. Therefore, he opined it was medically reasonable to conclude the miner had occupational pneumoconiosis that arose from his employment in the coal mines. In addition, the miner had chronic obstructive lung disease, peripheral vascular insufficiency, allergic rhinitis, and chronic sinusitis.

The second medical report of Dr. Rasmussen is dated January 22, 1992, and appears at DX 41-30. He noted that he evaluated the miner on January 14, 1992. He reviewed the miner's occupational history and past medical history. He noted a smoking history of 1¼ packs per day for thirty-four (34) years and continuing. The miner complained of shortness of breath for twenty (20) years, productive cough with respiratory infections, wheezing on exertion, and some chest discomfort. Physical examination revealed moderately reduced breath sounds, occasional rales, and prolongation of expiratory phase with forced expiration. Vent function studies revealed minimal restrictive and moderate obstructive vent impairment. Arterial blood gases showed a moderate resting hypoxemia. Previous chest x-rays were consistent with pneumoconiosis. Dr. Rasmussen diagnosed the miner as having occupational pneumoconiosis, chronic obstructive lung disease, deflected nasal septum, chronic sinusitis, peripheral vascular insufficiency, and decreased visual acuity. He stated that the miner's respiratory impairment was due to coal mine dust exposure with its resultant pneumoconiosis and his cigarette smoking. Dr. Rasmussen concluded the miner was totally disabled based on the table for impairment of respiratory function studies.

The third medical report of Dr. Rasmussen is dated January 22, 1992, and appears at DX 41-31. Dr. Rasmussen reviewed additional medical records. He agreed with Drs. Zaldivar, Fino, Tuteur, and Chillag that the miner had a disabling respiratory impairment that would prevent him form performing his last coal mine employment. Dr. Rasmussen noted that the opinions of others (Drs. Zaldivar, Fino, Tuteur, and Chillag), that coal mine dust exposure does not produce chronic obstructive lung disease, was not consistent with a growing body of medical opinions that indicated coal mine dust itself may produce significant and disabling chronic obstructive lung disease, even in the absence of cigarette smoking. He went on to discuss various studies that supported the relationship between obstructive lung disease and coal mine dust exposure.

Dr. Rasmussen concluded that the current medical literature supported the position that coal mine dust exposure could produce significant and disabling respiratory insufficiency both as a consequence of chronic bronchitis and/or emphysema. In addition, he noted that there was no way to separate the effects of cigarette smoking from those of coal mine dust exposure. Dr. Rasmussen noted the miner had findings consistent with emphysema. Based on the objective diagnostic studies, x-rays, and the miner's long coal mine dust exposure, Dr. Rasmussen concluded that the miner had coal workers' pneumoconiosis, which arose from his coal mine employment. In addition, the miner had both chronic bronchitis and emphysema, which were, at least in part, the consequence of coal mine dust exposure. Dr. Rasmussen acknowledged the miner had a significant smoking history which undoubtedly contributed to the miner's totally disabling respiratory insufficiency. He again noted there was no way to separate these two effects. Accordingly, Dr. Rasmussen concluded that the miner's coal mine dust exposure with its resultant pneumoconiosis was at least a major contributing factor to his totally disabling respiratory insufficiency.

Dr. Gregory Fino

The medical report of Dr. Fino is dated March 8, 1991, and appears at DX 41-38. Dr. Fino is Board-Certified in Internal Medicine and Pulmonary Disease. Dr. Fino conducted a medical records review at the Employer's request. He reviewed the miner's occupational history and noted at least forty-one (41) years of coal mine employment, ending in 1988. He summarized the medical evidence that had been developed to date. Dr. Fino opined the miner did not have simple coal workers' pneumoconiosis, based on the clinical evidence available. He noted the miner had a primary airway disorder which was not consistent with the abnormalities associated with pneumoconiosis and his chest x-ray did not show pneumoconiosis. Therefore, Dr Fino concluded the miner did not have CWP. Moreover, Dr. Fino opined the miner did not have industrial bronchitis because generally, once a miner left the mines, the affect of coal dust on the breathing tubes dissipated. More importantly, the miner's obstructive defect was a mix of large and small airways obstruction, which was not seen in industrial bronchitis. He noted that this pattern was classic for asthma or asthmatic bronchitis. Dr. Fino opined that the asthmatic bronchitis accounted for the miner's ventilatory impairment. He agreed with Dr. Zaldivar that the miner would be disabled, based on the most recent pulmonary function studies.

The second medical report of Dr. Fino is dated February 6, 1992, and appears at DX 41-46. Dr. Fino reviewed and summarized additional medical evidence, including the reports of Dr. Rasmussen. Dr. Fino opined that the additional information would not cause him to change his opinions. He stated that the miner had non-occupational asthma, asthmatic bronchitis, and hyperactive airway disease. Dr. Fino opined the miner would have had the same degree of pulmonary impairment had he never stepped foot in the mines.

Dr. Peter G. Tuteur

The medical report of Dr. Tuteur is dated April 6, 1991, and appears at DX 41-40. Dr. Tuteur is Board-Certified in Internal Medicine and Pulmonary Disease. Dr. Tuteur conducted a medical records review at the request of Employer. After summarizing the evidence, Dr. Tuteur

concluded there was insufficient objective data to justify a diagnosis of "clinically significant, physiologically significant, or radiographically significant" coal workers' pneumoconiosis. He noted that the miner did have a respiratory impairment in the form of a moderate obstructive vent defect not associated with a restrictive component and not associated with persistent impairment of gas exchange. He concluded that this impairment was due solely to cigarette-smoke-induced chronic obstructive pulmonary disease. Dr. Tuteur opined that the miner was totally disabled from his regular coal mine work, but that this disability was not caused in any way by coal workers' pneumoconiosis or the inhalation of coal dust.

Dr. Shawn Chillag

The medical report of Dr. Chillag is dated April 9, 1991, and appears at DX 41-40. Dr. Chillag is Board-Certified in Internal Medicine. Dr. Chillag performed a medical records review at the request of Employer. He noted the miner had an occupational history of forty-two (42) years of coal mine employment, retiring in 1988. He noted the miner had a varied smoking history of one (1) to two (2) packs of cigarettes per day from 27 to 40 years. Dr. Chillag concluded there was insufficient evidence to justify a diagnosis of coal workers' pneumoconiosis. He added that the miner's respiratory impairment was connected to COPD, and related to cigarette smoking and/or chronic asthma, but in no way was related to pneumoconiosis. Dr. Chillag opined the miner was totally disabled from his last coal mine employment, but that none of the disability was related to pneumoconiosis.

Dr. Robert Stanley

The medical note of Dr. Stanley is dated June 20, 1991, and appears at DX 41-25. Dr. Stanley noted that he had been treating the miner for the last 21 months for his severe respiratory condition. He also treated the miner for pneumonia on several occasions. He stated that the miner suffered from pneumoconiosis that had been demonstrated on chest x-ray showing chronic interstitial changes. Dr. Stanley noted the miner had been suffering from acute bronchitis, which he felt was directly related to his lung disease. He opined the miner could not return to his coal mine employment and that the miner was a "respiratory invalid," and would never be able to be employed.

Dr. A. Dahhan

The medical report of Dr. Dahhan is dated February 5, 1992, and appears at DX 41-46. Dr. Dahhan is Board-Certified in Internal Medicine and Pulmonary Disease and is a B-reader of chest x-rays. Dr. Dahhan conducted a medical records review at the request of Employer. He summarized the medical evidence developed to date. Dr. Dahhan concluded there was insufficient evidence for a diagnosis of occupational pneumoconiosis based on numerous negative readings of chest x-rays, the finding of an obstructive defect, and the finding of alteration of blood gas exchange mechanism. He added that the miner had a lengthy smoking history, sufficient in duration and amount to cause the development of chronic obstructive lung disease. Dr. Dahhan noted the miner had bronchial asthma which was genetic in nature. Dr. Dahhan opined the miner's respiratory capacity was impaired to a degree that he was unable to return to his previous coal mine employment. He concluded the miner's pulmonary disability

was the result of chronic bronchitis and possible emphysema and asthma, all conditions of the general public at-large, and not caused by, or related to, coal mine employment or the inhalation of coal mine dust. Dr. Dahhan noted that even if the miner had simple pneumoconiosis, the amount of airways obstruction that was seen was out of proportion to what one would expect. This further reinforced his opinion that the miner's respiratory impairment was not the result of occupational exposure.

E. Death Certificate

The death certificate is dated May 20, 1997, and appears at DX 11. The date of the miner's death is listed as May 16, 1997. The certifying physician was Dr. Robert Stanley. The immediate causes of death were noted as cardiopulmonary arrest, congestive heart failure, laryngeal cancer, and atrial fibrillation. No autopsy was performed.

F. Post-Mortem Reports

Dr. D.L. Rasmussen

The medical report of Dr. Rasmussen is dated June 23, 1998, and appears at DX 24. Dr. Rasmussen reviewed the medical evidence and noted the miner had a history of 40 years of coal mine employment, ending in 1988. Most of the miner's work was at the face of the mine. Dr. Rasmussen noted that the record included both positive and negative x-ray interpretations. He noted the miner showed signs of a progressive impairment in pulmonary function as early as February 1984. Dr. Rasmussen indicated the miner had experienced a progressive loss in pulmonary function in his most recent vent study in March 1998.

Most of the reviewers had agreed that the miner had suffered from a totally disabling respiratory impairment, but disagreed as to the cause. Most of the other reviewers attributed the miner's impairment, at least in part, to cigarette smoking. Dr. Rasmussen went on to discuss the relationship between coal mine dust and the development of chronic obstructive pulmonary disease. He also pointed out that in Germany and Great Britain, chronic bronchitis and emphysema were considered occupationally related to coal mine dust exposure. Dr. Rasmussen noted that the miner had a progressively severe impairment in pulmonary function, and required hospitalization on several occasions for respiratory failure with increased dyspnea, cough, wheeze, and hypoxemia. He was in very severe respiratory distress at the time of his last hospitalization. Dr. Rasmussen acknowledged the miner suffered from carcinoma of the larynx, pneumonitis, perforated colon with subsequent peritonitis, and septicemia. Dr. Rasmussen opined that it was clear that the miner's chronic obstructive lung disease was a major contributing factor to his death. He concluded the chronic obstructive lung disease was a consequence of his previous smoking and coal mine dust exposure. He added the miner's coal mine dust exposure, with its resultant occupational pneumoconiosis, was a material contributing factor to the miner's death.

Dr. Alan Ducatman

The medical report of Dr. Ducatman is dated October 28, 1998, and appears at DX 24. Dr. Ducatman reviewed the miner's occupational history and noted a smoking history of one (1) to two (2) packs of cigarettes per day for 33-40 years. He noted an occupational history of forty (40) years of coal mine employment and that the miner had substantial exposure to dust. He noted that earliest records documented the presence of parenchymal fibrosis as early as 1983, and that the miner had been dyspneic with exertion since 1981. Dr. Ducatman stated that, although many of the miner's vent studies were interpreted as showing an obstructive defect, the studies actually were a mixed restrictive/obstructive defect. He concluded that the overall picture appeared to include exposure to asbestos as well as coal dust. Dr. Ducatman concluded the miner died of complications from laryngeal cancer and, had this not caused his death, the miner would have died of advanced respiratory disease. Dr. Ducatman diagnosed the miner with the following:

1. Chronic obstructive/restrictive pulmonary disease with chronic COPD/chronic bronchitis due to cigarette smoking, coal workers' pneumoconiosis, and asbestosis.
2. Terminal Laryngeal Cancer due to cigarette smoking, asbestos exposure, diesel exhaust, and alcohol (?).
3. Coronary artery disease/peripheral vascular disease/(? Congestive heart failure in extremis) due to cigarette smoking and other risk factors.
4. Bowel perforation/septicemia in extremis due to aspiration pneumonitis (due to laryngeal cancer) and pulmonary/vascular compromise.
5. Coal Workers' Pneumoconiosis due to mine dust and cigarette smoking.
6. Asbestosis due to asbestos dust (source not characterized in available record) and cigarette smoking.
7. Parkinson's Disease
8. Gouty Arthritis
9. Hypothyroidism
10. Non-Insulin Dependent Diabetes Mellitus

Dr. Ducatman concluded the miner died of late complications of laryngeal cancer and had he not died of laryngeal cancer, he was near death from respiratory disease. The miner also had radiographic evidence of asbestos exposure. He noted that although cigarette smoking was the cause of laryngeal cancer, asbestos exposure was also a contributing cause. He therefore concluded that the miner's terminal cancer could be attributed, in part, to his asbestos exposure.

The deposition testimony of Dr. Ducatman is dated June 14, 2001, and appears at EX 8. The deposition was taken by Employer without counsel or a representative of Claimant present. Dr. Ducatman was Board-Certified in Internal Medicine and Occupational Medicine. He acknowledged the miner had significant exposure to coal mine dust and had a significant history of smoking. He noted that there was no history of asbestos exposure in the records. He felt that the description of pleural plaques was consistent with a diagnosis of asbestosis. He disagreed that there was a dispute among the x-ray interpretations. He noted that some readers look only for changes associated with coal workers' pneumoconiosis and do not focus on pleural changes

consistent with asbestosis. Dr. Ducatman opined the miner had a mixed restrictive/obstructive defect, but admitted that the February 1983 study showed only an obstructive defect.

Without the medical records from Mt. Carmel Medical Center, where the miner was treated from April 1997, Dr. Ducatman was forced to speculate regarding the cause of death. He did not know whether the miner had significant congestive heart failure prior to his death. He stated, regarding the etiology of the miner's laryngeal cancer, "I speculate --I do have to tell you that at this point, it has to be speculation --that asbestosis is also a contributing factor." He acknowledged he had not personally reviewed the x-rays, so there was some question as to whether they were really consistent with a pattern of asbestosis. Dr. Ducatman also acknowledged there was a dispute in the medical literature as to whether or not there was a link between asbestos exposure and the development of laryngeal cancer. He also admitted he had no history that the miner was exposed to diesel fumes. He admitted coal workers' pneumoconiosis did not cause laryngeal cancer. Dr. Ducatman opined that coal workers' pneumoconiosis did not play a role in causing the miner's death.

Dr. Mark Bates

The medical report of Dr. Bates is undated, and appears at DX 30. He stated there was "no question that this patient's black lung disease hastened his demise." He noted that the miner had end stage COPD and coal workers' pneumoconiosis that had been documented by his pulmonary team, led by Dr. D'Brot. He added that the miner had developed COPD-related cachexia and that this clearly placed him in a weakened condition, which ultimately hastened his demise. He noted that patients with end stage lung disease can develop wasting protein loss and a weakened state. He opined the miner had all of these qualities and his cachexia made it very difficult to tolerate the cardiac and pulmonary complications that lead to his demise. Dr. Bates stated, "I personally feel that his arrest situation was a primary pulmonary event and potentially related to hypoxia, decompensation, etc."

Dr. Gregory Fino

The medical report of Dr. Fino is dated June 14, 2001, and appears at EX 4. Dr. Fino reviewed and summarized the extensive medical records in this matter. Dr. Fino stated that the miner had numerous problems related to smoking and that he had significant COPD, as evidenced on the vent studies. He noted the miner had intermittent hypoxemia which was more consistent with cigarette smoking than pneumoconiosis. He added that the miner developed cancer of the voice box, which was directly related to cigarette smoking. He noted that he saw no evidence of a coal mine dust-related pulmonary condition, but that his treatment record was for a lung disease consistent with a smoking-related condition. Dr. Fino pointed out that the physician who signed the death certificate did not mention any lung disease diagnoses. Dr. Fino acknowledged the miner had a serious lung disease, but that he also had other medical problems, including cancer of the larynx, a possible lung cancer, significant coronary artery disease with previous heart attacks and irregular heart beats, and a perforated colon. Dr. Fino stated:

> Because of the multitude of medical problems, it is pure speculation to suggest that lung disease, regardless of etiology was a contributing cause of death. Since

> no autopsy was performed, and since he died at home, how do we know that he did not have a massive stroke, a ruptured abdominal aneurysm, or another heart attack?

He also noted that even if the miner died due to lung disease, the lung disease was related to cigarette smoking. Dr. Fino stated the following conclusions:

1. no pneumoconiosis was present
2. a disabling respiratory impairment was present due to cigarette smoking
3. coal mine dust inhalation did not cause or contribute to his disability
4. based on the available data, the miner's death was neither caused in whole nor in part by lung disease
5. even if lung disease played some role in his death, the lung disease due to coal mine dust inhalation neither caused, contributed to, nor hastened death
6. the miner would have died as and when he did had he never stepped foot in the mines, there is no evidence that lung disease due to coal mine dust inhalation played a role in the miner's death

The deposition of Dr. Fino is dated September 7, 2001, and appears at EX 9. The deposition was taken by Employer and no representative of Claimant was present. Dr. Fino reviewed x-rays, CT scans, and pulmonary function study results. Dr. Fino noted the miner had no restrictive defect, based on lung volumes, which was the "gold standard." He disagreed with Dr. Bates' conclusion regarding cause of death because there was no medical information at or about the time of death. The last medical record was three weeks before the miner's death. He noted that lung disease may have played a role in death, but that was pure speculation. He stated that it would not be medically reasonable to unquestionably state that the miner's death was related to coal dust, when the cause of his death was not known. Dr. Fino noted that he did not believe that the miner had a coal mine dust-induced lung disease. He stated that the pattern and severity of the obstruction was "much more consistent with smoking, and the worsening of his lung functioning over time was much more consistent with smoking than it was a coal dust related condition." Dr. Fino opined that he could not assess whether or not lung disease played a role in causing the miner's death because there was no objective evidence as to what caused the miner's death. He added that even if the miner had a chronic dust disease, and assuming lung disease played a role in the miner's death, the affect on the miner's death would have been negligible.

Dr. George L. Zaldivar

The deposition of Dr. Zaldivar was taken on August 21, 2001, and appears at EX 10. The deposition was taken by Employer and no representative of Claimant was present. Dr. Zaldivar agreed that coal mine dust exposure could cause obstructive lung disease and that one did not need a positive chest x-ray to diagnose pneumoconiosis. He stated that the miner had a purely obstructive defect with no restriction, based on lung volumes. He opined the miner's lung disease was due to asthma and smoking-induced emphysema. He reviewed a series of chest x-rays and found no evidence of pneumoconiosis or asbestosis. He noted the presence of bullae caused typically by smoking. The miner also had indications of congestive heart failure on x-ray. Dr. Zaldivar noted that there was no evidence of dust retention in the lungs; therefore, one

would not expect to find any clinical evidence of coal workers' pneumoconiosis, such as an airway obstruction. Dr. Zaldivar noted there was no causal link between coal mine dust exposure and laryngeal cancer. He added there was absolutely no reason to speculate about asbestos causing any problem with this cancer. Dr. Zaldivar acknowledged the miner had significant cardiac disease and lung disease. He concluded there was no coal mine dust-induced lung disease in this case, and that the miner had a pulmonary impairment caused by significant smoking and underlying asthma. He opined the miner's death was not in any way hastened, aggravated, or caused by a coal mine dust-induced lung disease.

Dr. James R. Castle

The medical report of Dr. Castle is dated July 2, 2002, and appears at EX 11. Dr. Castle is Board-Certified in Internal Medicine and Pulmonary Disease. He reviewed and summarized the medical evidence of record. He concluded the miner did not suffer from pneumoconiosis. He noted the miner had two risk factors for the development of respiratory symptoms: coal mine dust and smoking. He noted the miner also had evidence of asthma, coronary artery disease, and congestive heart failure. Dr. Castle added that the miner developed laryngeal cancer due to cigarette smoking. He concluded there was no radiographic evidence of pneumoconiosis based on the interpretations of the vast majority of radiologists and B-readers. He noted that the CT scans also did not show changes of pneumoconiosis.

Dr. Castle acknowledged the miner had progressive airways obstruction, which, on some occasions, demonstrated a significant degree of reversibility. He added the miner did not have any restrictive defect. The miner had a significant reduction in diffusing capacity, indicating the development of smoking induced emphysema. Dr. Castle stated that coal workers' pneumoconiosis did not normally cause a reduction in the diffusing capacity (indicative of emphysema) and when it did occur, it occurred in the presence of a high degree of "p" or "r" type opacities on the chest x-ray. These changes were not seen on the chest x-rays. He also noted that the varying degrees of hypoxemia were not typical of pneumoconiosis because in that circumstance, the degree of hypoxemia was constant and did not improve after exercise. Dr. Castle concluded the miner did not have the physical findings, the radiographic findings, the physiologic findings, or the arterial blood gas findings to indicate the presence of pneumoconiosis. He opined the miner's death was due to complications of a ruptured diverticulum with sepsis syndrome. He added that even if death were due to complications of laryngeal cancer, this would have no relationship to coal mine dust exposure. Dr. Castle opined the miner's death was not the result of coal workers' pneumoconiosis, and his death was neither caused by, contributed to, or hastened in any way by coal mine dust-induced lung disease. He added that even if the miner had simple coal workers' pneumoconiosis, his opinion regarding the cause of death would not change. He stated that none of the findings around the miner's death were related to any lung disease; they were instead due to a ruptured diverticulum in the sigmoid colon with resultant sepsis syndrome and complications thereof.

The deposition of Dr. Castle is dated November 12, 2002, and appears at EX 17. He stated that the vent studies showed a significant degree of reversible airway obstruction and pulmonary emphysema that indicated the presence of both tobacco smoke-induced airway obstruction and bronchial asthma. He noted that patients with coal workers' pneumoconiosis

generally had a mixed irreversible obstruction and restriction. In this case, he noted a significant degree of reversibility and no restriction. He further noted the arterial blood gases showed varying degrees of hypoxemia, which was atypical for coal workers' pneumoconiosis. He opined the miner died due to diverticular disease of the colon. He opined the miner would have died in the same manner and time, whether or not he worked in the mines or whether or not he smoked cigarettes. He disagreed with Dr. Rasmussen's conclusion that COPD was a major contributing factor to the miner's death. He noted that ruptured sepsis occurred in people, regardless of their occupational history. He also disagreed with the opinion of Dr. Bates. He agreed that the miner probably had cachexia and protein loss, but as a result of the sepsis syndrome, and it had nothing to do with pulmonary circumstances. He added that he did not believe that the primary cause of death was cardiac or pulmonary. He stated that the miner did not have legal or medical pneumoconiosis. Dr. Castle admitted coal dust exposure could cause COPD, emphysema, and industrial bronchitis. He likewise admitted the miner had a totally disabling respiratory impairment. When asked whether a disabling pulmonary impairment would effect the body's ability to deal with bacteriologic infections, Dr. Castle stated, "No, not necessarily. If -- it would have if one had developed a primary pneumonia…then yes, that would have an impact." He then acknowledged that Dr. Rasmussen may have noted the presence of pneumonia. He then stated that it was impossible to know whether the miner had pneumonia around the time of death without an x-ray.

Dr. Ben V. Branscomb

The medical report of Dr. Branscomb is dated October 19, 2002, and appears at EX 12. Dr. Branscomb is Board-Certified in Internal Medicine. Dr. Branscomb reviewed nearly one thousand pages of documents related to this matter. He noted an occupational exposure to coal mine dust sufficient to acquire coal workers' pneumoconiosis in a susceptible individual. He also noted a "devastating" smoking history of about two (2) packs per day for at least 30 years. He concluded the x-ray evidence overwhelmingly was negative for coal workers' pneumoconiosis. There was not sufficient objective evidence to justify a diagnosis of coal workers' pneumoconiosis. He added the miner was totally disabled by respiratory disease, beginning about 1989 or 1990. The respiratory disease consisted of asthmatic bronchitis, COPD with acute asthmatic type, and hyperactivity or asthmatic exacerbations. He noted that the exacerbations were caused by episodes of respiratory infection and that the cause of this disease was very heavy cigarette smoking in a person with a tendency to bronchospasm. He added that the miner's chronic asthmatic bronchitis did not differ from the disease found in the population generally. Dr. Branscomb concluded the miner was totally disabled from performing his last coal mine employment prior to his death due to his respiratory insufficiency. He added that coal dust exposure played no role in any of the miner's impairment. It did not cause any of his diseases nor did it aggravate or predispose to any of his diseases or impairments. He noted that even if the miner had coal workers' pneumoconiosis, it would still be his judgment that coal workers' pneumoconiosis played no role in his impairment, no role in the cancer, and nor role in causing or accelerating death.

G. Other Medical Evidence

Occupational Pneumoconiosis Board

The record contains the findings of the Occupational Pneumoconiosis Board. On February 21, 1984, the board found that the miner had occupational pneumoconiosis with no functional impairment attributable. He was granted a statutory 5% award. (DX 41-3). On May 22, 1992, the board issued a decision that the miner had occupational pneumoconiosis with no functional impairment and that the miner had been paid a statutory award of 5%. (DX 24). On July 21, 1998, the board issued a decision that occupational pneumoconiosis was not a materially contributing factor to death. (DX 24). Claimant protested the decision and the matter was presumably settled by the parties. (DX 25).

Hospital Records

The record also contains the medical records from an admission to Dunlap Memorial Hospital from September 21, 1989 to September 24, 1989, with a diagnosis of asthmatic bronchitis, history of black lung disease, COPD, and a smoking history of 2 packs per day for 40 years. (DX 41-11). There are also records for various admissions to Summersville Memorial Hospital from March 1994 through April 1996. During that time, the miner was treated extensively for shortness of breath, acute exacerbation of COPD, and acute bronchitis. (DX 37). The miner was also treated at Plateau Medical Center throughout 1996 for exacerbation of COPD, congestive heart failure, acute bronchopneumonia, pneumonia, pneumoconiosis, and coronary artery disease. (DX 39). The miner was also treated at Charleston Medical Center in July and August 1996, for pneumonia, diarrhea, COPD, laryngeal cancer, CAD, and congestive heart failure. (DX 13).

From January through April 1997, the miner was treated for pneumonia, end stage lung disease, CAD, coal workers' pneumoconiosis, COPD, and pneumonic infiltrate. (DX 12, 13, 39, and 24). The miner was transferred to Mt. Carmel Medical Center where the last note was dated May 13, 1997. (DX 24). The miner was released to go home, and on May 16, 1997, the miner died. (DX 11).

CONCLUSIONS OF LAW

Length of Coal Mine Employment

In the miner's claim for benefits, the parties stipulated to forty (40) years of coal mine employment. (DX 41-64). The District Director, in the instant matter, found the record of earnings from the Social Security Administration and a letter from Employer showed that the miner worked 40.01 years in the coal mines. (DX 32). Nonetheless, in its pre-hearing report (ALJ-1) and in Form CM-1025 (DX 42), Employer noted it was contesting the length of coal mine employment. In its closing brief, Employer did not address this issue.

I find that the evidence of record, specifically the statement of work history included with the application for benefits (DX 2), the September 27, 1988 letter from Employer (DX 3), and the miner's social security earnings (DX 5), supports the District Director's finding of 40.01 years of coal mine employment. No evidence to the contrary has been produced by Employer. Accordingly, I find Claimant has established 40.01 years of qualifying coal mine employment.

Date of Filing

I find that Claimant filed her claim for benefits under the Act on February 9, 1999. (DX 1).

Responsible Operator

I find that Island Creek Coal Company is the responsible operator and will provide payment of any benefits awarded to Claimant. (DX 32; DX 3).

Dependents

I find Claimant was married to the miner on June 22, 1968. (DX 8). The miner's death certificate indicated they were still married at the time of the miner's death. (DX 11). In her application for benefits, Claimant indicated she was the miner's surviving spouse. (DX 1). Claimant did not claim to have any other dependents for purposes of augmentation of benefits under the Act. (DX 1). Accordingly, I find Claimant is an eligible survivor of the miner and has no dependents for purposes of augmentation of benefits under the Act.

Entitlement to Benefits

Applicable Regulations

Claimant's claim for benefits was filed on February 9, 1999, and is governed by the Part 718 Regulations. However, on January 19, 2001, substantial changes to Parts 725 and 718 of the Federal Regulations became effective. (*See* footnote 1). Based upon my review of the new Regulations, there are two sections that specifically deal with the question of whether these new Regulations are applicable to cases that are currently pending at the time of the enactment.

Pursuant to § 725.2, the revisions of Part 725 shall also apply to the adjudication of claims that were pending on January 19, 2001, except for the sections enumerated in 20 C.F.R. § 725.2(c). Accordingly, with the exception of those sections listed as an exemption, the revisions to Part 725 will apply to the facts of this decision.

Pursuant to § 718.101, the standards for the administration of clinical tests and examinations contained in subpart B "shall apply to all evidence developed by any party after January 19, 2001 in connection with a claim governed by this part...." 20 C.F.R. § 718.101(b) (emphasis added). Accordingly, since the evidence in the instant matter was developed prior to January 19, 2001, the newly enacted § 718, subpart B does not apply.

On August 9, 2001, U.S. District Court Judge Emmet Sullivan upheld the validity of the new Regulations in *National Mining Association v. Chao*, No. 00-3086 (D.D.C. Aug. 9, 2001). However, on June 14, 2002, the United States Court of Appeals for the District of Columbia Circuit ("the court") affirmed in part, reversed in part, and remanded the case. *See National Mining Association v. Department of Labor*, No. 01-5278 (June 14, 2002). Accordingly, I will apply the sections of the newly revised version of Part 718 (*i.e.* subparts A, C and D) and Part 725 that took effect on January 19, 2001, that the court did not find impermissibly retroactive to the facts of the instant matter. (*See* footnote 1).

Standard of Review

The administrative law judge need not accept the opinion of any particular medical witness or expert, but must weigh all the evidence and draw his/her own conclusions and inferences. *Lafferty v. Cannelton Industries, Inc.*, 12 B.L.R. 1-190 (1989); *Stark v. Director, OWCP*, 9 B.L.R. 1-36 (1986); *Todd Shipyards Corp. v. Donovan*, 300 F.2d 741 (5th Cir. 1962). The adjudicator's function is to resolve the conflicts in the medical evidence; those findings will not be disturbed on appeal if supported by substantial evidence. *Lafferty, supra; Fagg v. Amax Coal Co.*, 12 B.L.R. 1-77 (1988), *aff'd*, 865 F.2d 916 (7th Cir. 1989);*Short v. Westmoreland Coal Co.*, 10 B.L.R. 1-127 (1987); *Piccin v. Director, OWCP*, 6 B.L.R. 1-616 (1983). *Peabody Coal Co. v. Lowis*, 708 F.2d 266, 5 B.L.R. 2-84 (7th Cir. 1983).

In considering the medical evidence of record, an administrative law judge must not selectively analyze the evidence. See *Wright v. Director, OWCP*, 7 B.L.R. 1-475 (1984); *Hess v. Clinchfield Coal Co.*, 7 B.L.R. 1-295 (1984); *Crider v. Dean Jones Coal Co.*, 6 BLR 1-606 (1983); *Peabody Coal Co. v. Lowis*, 708 F.2d 266, 5 B.L.R. 2-84 (7th Cir. 1983); *see also Stevenson v. Windsor Power House Coal Co.*, 6 B.L.R. 1-1315 (1984). Determinations concerning the weight of the evidence, and the credibility of medical experts and witnesses, however, are for the administrative law judge. *Mabe v. Bishop CoalCo.*, 9 B.L.R. 1-67 (1986); *Brown v. Director, OWCP*, 7 B.L.R. 1-730 (1985); see also *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985); *Henning v. Peabody Coal Co.*, 7 B.L.R. 1-753 (1985);*Peabody Coal Co. v. Benefits Review Board*, 560 F.2d 797, 1 B.L.R. 2-133 (7th Cir. 1977).

As the trier-of-fact, the administrative law judge has broad discretion to assess the evidence of record and determine whether a party has met its burden of proof.

Kuchwara v Director, OWCP, 7 B.L.R. 1-167 (1984). In considering the evidence on any particular issue, the administrative law judge must be cognizant of which party bears the burden of proof. Claimant has the general burden of establishing entitlement and the initial burden of going forward with the evidence. See *White v. Director, OWCP*, 6 B.L.R. 1-368 (1983).

Entitlement: In General

To establish entitlement to survivor's benefits, a claimant must establish that the miner had pneumoconiosis, that the miner's pneumoconiosis arose out of coal mine employment, and that the miner's death was due to pneumoconiosis. 20 C.F.R. §§718.3, 718.202, 718.203, 718.205(a); *Trumbo v. Reading Anthracite Co.*, 17 B.L.R. 1-85 (1993); *Haduck v. Director, OWCP*, 14 B.L.R. 1-29 (1990); *Neeley v. Director, OWCP*, 11 B.L.R. 1-85 (1988); *Boyd v. Director, OWCP*, 11 B.L.R. 1-39 (1988). For a survivor's claims filed on or after January 1, 1982, the miner's death will be considered due to pneumoconiosis if pneumoconiosis was the cause of the miner's death, was a substantially contributing cause or factor leading to the miner's death, death was caused by complications of pneumoconiosis or the presumption, relating to complicated pneumoconiosis, set forth at Section 718.304, is applicable. 20 C.F.R. §718.205(c)(1)-(3). Pneumoconiosis is a substantially contributing cause of death if it hastened the miner's death. 20 C.F.R. §718.205(c)(5); *Shuff v. Cedar Coal Co.*, 967 F.2d 977, 16 B.L.R. 2-90 (4th Cir. 1992), *cert. denied*, 506 U.S. 1050 (1993).

The Existence of Pneumoconiosis and the Application of Collateral Estoppel

As noted previously, Administrative Law Judge Feirtag found, in the living miner's claim, the miner suffered from pneumoconiosis, but ultimately denied benefits. The miner appealed the denial of benefits and Employer cross-appealed regarding the finding of the existence of pneumoconiosis. The Benefits Review Board affirmed the administrative law judge's decision in its entirety. No further action was taken on the claim.

In the instant survivor's claim, Employer contested the issue of the existence of pneumoconiosis. This raises the threshold issue of whether Employer is collaterally estopped from re-litigating the existence of coal worker's pneumoconiosis in a survivor's claim where the miner was found to have pneumoconiosis in his living miner's claim.[7]

For collateral estoppel to apply in the present case, which arises within the jurisdiction of the United States Court of Appeals for the Fourth Circuit, Claimant must establish that:

[7] Both parties failed to raise and address the issue of collateral estoppel in their closing briefs. However, the courts have generally held that the adjudicator may raise the issue of collateral estoppel *sua sponte*. In *Studio Art Theatre of Evansville, Inc. v. City of Evansville*, 76 F.3d 128, 130 (7th Cir. 1996), the Circuit Court held that the benefits of precluding re-litigation of issues runs not only to the litigants, but to the judicial system. Moreover, in *Doe v. Pfrommer*, 148 F.3d 73 (2d Cir. 1998), it was held that "strong public policy in economizing the use of judicial resources by avoiding re-litigation "favors *sua sponte* application of collateral estoppel." *See also Tri-Med Finance Co. v. National Century Finance Enterprises, Inc.*, 208 F.3d 215 (6th Cir. 2000).

(1) the issue sought to be litigated is identical to the one previously litigated;
(2) the issue was actually determined in the prior proceeding;
(3) the issue was a critical and necessary part of the judgment in the prior proceeding;
(4) the prior judgment is final and valid; and
(5) the party against whom the estoppel is asserted had a full and fair opportunity to litigate the issue in the previous forum.

See Sedlack v. Braswell Services Group, Inc., 134 F.3d 219 (4th Cir. 1998); *Hughes v. Clinchfield Coal Co.*, 21 B.L.R. 1-134 (1999)(*en banc*).

At the time of the adjudication of the miner's claim, evidence sufficient to establish pneumoconiosis under one of the four methods set out at 20 C.F.R. §718.202(a)(1)-(4) obviated the need to do so under any of the other methods. *See Dixon v. North Camp Coal Co.*, 8 B.L.R. 1-344 (1985). However, subsequent to the issuance of the award of benefits in the miner's claim, the Fourth Circuit held that although Section 718.202(a) enumerates four distinct methods of establishing pneumoconiosis, all types of relevant evidence must be weighed together to determine whether a miner suffers from the disease. *See Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000); *see also Penn Allegheny Coal Co. v. Williams*, 114 F.3d 22, 21 B .L.R. 2-104 (3d Cir. 1997). In light of the change in law enunciated in *Compton*, which overruled the Board's holding in *Dixon*, the issue of whether the existence of pneumoconiosis has been established pursuant to Section 718.202(a) is not identical to the one previously litigated and actually determined in the miner's claim. *See Sedlack, supra*; *Hughes, supra*. Thus, inasmuch as each of the prerequisites for application of the doctrine of collateral estoppel is not present, I find that the doctrine of collateral estoppel is not applicable in this survivor's claim regarding the existence of pneumoconiosis pursuant to 20 C.F.R. §718.202(a). *See Surway v. United Pocahontas Coal Co*, BRB No. 01-0881 BLA (June 26, 2002)(unpub.); *Howard v. Valley Camp Coal Co.*, BRB No. 001034 BLA (August 24, 2001)(unpub.); *Price v. Consolidated Coal Co.*, BRB No. 00-0453 BLA (January 24, 2001)(unpub.). Consequently, I will reconsider the evidence and determine whether it is sufficient to establish the existence of pneumoconiosis in accordance with the standard enunciated in *Compton.*[8]

[8] The instant case is distinguishable from the recent case of *Zeigler Coal Co. v. Director, OWCP [Villain]*, 312 F.3d 332 (7th Cir. 2002). In that case, the Seventh Circuit held that an employer is collaterally estopped from re-litigating the existence of pneumoconiosis in a survivor's claim where the miner was awarded benefits based on a lifetime claim and no autopsy evidence is presented in the survivor's claim. The claimant there only had to prove the existence of pneumoconiosis within one of the discrete subsections of §718.202(a). After the underlying case in *Zeigler* was decided (on December 7, 1999), the Fourth Circuit adopted the new standard enunciated in *Compton, supra*. The Seventh Circuit has not yet ruled on this issue of weighing evidence together under §718.202(a)(1)-(4). Therefore, since there has not been any change in the law in the Seventh Circuit regarding the weighing of the evidence under this subsection, the issue in the survivor's claim was identical to the issue in the living miner's claim and collateral estoppel could be applied. Conversely, in the instant case, pursuant to the Fourth Circuit's recent holding in *Compton, supra*, the issue now presented in the survivor's claim is not identical to the

I. The Existence of Pneumoconiosis

Thirty U.S.C. § 902(b) and 20 C.F.R. § 718.201 define pneumoconiosis as "a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment."[9] The definition is not confined to "coal workers' pneumoconiosis," but also includes other diseases arising out of coal mine employment, such as anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, progressive massive fibrosis, silicosis, or silicotuberculosis.[10] 20 C.F.R. § 718.201. The term "arising out of coal mine employment" is defined as including "any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust

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issue that was presented in the living miner's claim in 1984. Therefore, since all of the prerequisites for the application of collateral estoppel are not present, the doctrine of collateral estoppel is not applicable to the existence of pneumoconiosis issue in the instant survivor's claim.

[9] Pneumoconiosis is a progressive and irreversible disease; once present, it does not go away. *Mullins Coal Co. v. Director, OWCP*, 484 U.S. 135, 151 (1987); *Lisa Lee Mines v. Director*, 86 F.3d 1358 (4th Cir. 1996)(*en banc*) at 1364; *LaBelle Processing Co. v. Swarrow*, 72 F.3d 308 (3d Cir. 1995) at 314-315.

[10] Regulatory amendments, effective January 19, 2001, state:

> (a) For the purpose of the Act, "pneumoconiosis" means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical", pneumoconiosis and statutory, or "legal", pneumoconiosis.
>
> (1) *Clinical Pneumoconiosis*. "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconioses, i.e., the conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment.
>
> (2) *Legal Pneumoconiosis*. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.
>
> (b) For purposes of this section, a disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.
>
> (c) For purposes of this definition, "pneumoconiosis" is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.

20 C.F.R. § 718.201(a)(emphasis added).

exposure in coal mine employment."

"…[T]his broad definition 'effectively allows for the compensation of miners suffering from a variety of respiratory problems that may bear a relationship to their employment in the coal mines.'" *Robinson v. Pickands Mather & Co./Leslie Coal Co. & Director, OWCP*, 14 B.L.R. 2-68 (4th Cir. 1990) at 2-78, 914 F.2d 35 (4th Cir. 1990)(*citing, Rose v. Clinchfield Coal Co.*, 614 F. 2d 936, 938 (4th Cir. 1980)). Thus, asthma, asthmatic bronchitis, or emphysema may fall under the regulatory definition of pneumoconiosis if they are related to coal dust exposure. *Robinson v. Director, OWCP*, 3 B.L.R. 1-798.7 (1981); *Tokarcik v. Consolidation Coal Co.*, 6 B.L.R. 1-666 (1983). Likewise, chronic obstructive pulmonary disease may be encompassed within the legal definition of pneumoconiosis. *Warth v. Southern Ohio Coal Co.*, 60 F.3d 173 (4th Cir. 1995).

The claimant has the burden of proving the existence of pneumoconiosis by any one of four methods. The Regulations provide the means of establishing the existence of pneumoconiosis by: (1) a chest x-ray meeting the criteria set forth in 20 C.F.R. § 718.202(a); (2) a biopsy or autopsy conducted and reported in compliance with 20 C.F.R. § 718.106; (3) application of the irrefutable presumption for "complicated pneumoconiosis" found in 20 C.F.R. § 718.304; or (4) a determination of the existence of pneumoconiosis made by a physician exercising sound judgment, based upon certain clinical data, as well as medical and work histories, supported by a reasoned medical opinion. 20 C.F.R. § 718.202(a). Pulmonary function studies are not diagnostic of the presence or absence of pneumoconiosis. *Burke v. Director, OWCP*, 3 B.L.R. 1-410 (1981). As noted previously, in *Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000), the Fourth Circuit held that the administrative law judge must weigh all evidence together under 20 C.F.R. § 718.202(a) to determine whether the miner suffered from coal workers' pneumoconiosis.

A. Chest X-ray Evidence

A finding of the existence of pneumoconiosis may be made with positive chest x-ray evidence. 20 C.F.R. § 718.202(a)(1). The existence of pneumoconiosis may be established by chest x-rays classified as category 1, 2, 3, A, B, or C according to ILO-U/C International Classification of Radiographs standard. A chest x-ray classified as category 0, including subcategories 0/-, 0/0, 0/1, does not constitute evidence of pneumoconiosis. 20 C.F.R. § 718.102(b). Where two or more x-ray reports are in conflict, the radiologic qualifications of the physicians interpreting the x-rays must be considered. §718.201(a)(1).

While a judge is not required to defer to the numerical superiority of x-ray evidence, although it is within his or her discretion to do so. *Wilt v. Woverine Mining Co.*, 14 B.L.R. 1-70 (1990) citing *Edmiston v. F & R Coal*, 14 B.L.R. 1-65 (1990). The ALJ must rely on the evidence which he deems to be most probative, even where it is contrary to the numerical majority. *Tokarcik v. Consolidation Coal Co.*, 6 B.L.R. 1-666 (1984).

In addition, the Fourth Circuit noted that pneumoconiosis is "progressive and irreversible," such that it is proper to accord greater weight to later positive x-ray studies over earlier negative studies. It stated further that generally, "later evidence is more likely to show the

miner's current condition," where it is consistent in demonstrating a worsening of the miner's condition. *Lane Hollow Coal Co. v. Director, OWCP [Lockhart]*, 137 F.3d 799 (4th Cir. 1998).

Of the submitted evidence, there are ninety seven (97) interpretations of twenty-five (25) x-rays and two (2) CT scans in the record. I accord less weight to the interpretations of x-rays taken on 1-3-85 (DX 41-40), 10-30-86 (DX 41-40), 9-27-89 (DX 41-40), 9-29-89 (DX 41-40, DX 41-11), 10-3-89 (DX 41-40, DX 41-11), 3-14-94 (DX 37), 3-20-94 (DX 37), and 3-25-94 (DX 37). These x-rays were taken in the hospital for purposes of diagnosing acute medical conditions of the miner and not for purposes of diagnosing the presence of pneumoconiosis. I find that, given the setting in which these x-rays were taken and read, the omission of a finding of pneumoconiosis in this instance does not necessarily mean that the disease was not present. For this reason I accord these readings less weight. *See Sacolick v. Rushton Mining Co.*, 6 B.L.R. 1-930 (1984).

Of the remaining interpretations, there were sixty-three (63) readings by Board-Certified Radiologists and B-readers of seventeen (17) x-rays. The Board has held that it is proper to credit the interpretation of a dually qualified physician over the interpretation of a B-reader. *Cranor v. Peabody Coal Co.*, 22 BLR 1-1 (1999)(*en banc* on recon.). However, an administrative law judge may utilize any reasonable method of weighing such evidence. *See Sexton v. Director, OWCP*, 725 F.2d 213 (6th Cir. 1985). Of the sixty-three (63) readings, fifty-eight (58) interpretations were negative and five (5) were positive for pneumoconiosis.

Additionally, there were ten (10) readings by Board-Certified Radiologists and B-readers of two (2) CT scans. All ten readings were negative for pneumoconiosis.

Therefore, I find, based on the wealth of negative interpretations by dually-qualified physicians, that Claimant has failed to establish, by the preponderance of the evidence, the existence of pneumoconiosis pursuant to §718.202(a)(1).

B. Biopsy Evidence

A biopsy may be the basis for a finding of the existence of pneumoconiosis. §718.202(a)(2). A finding in a biopsy of anthracotic pigmentation, however, shall not be sufficient, by itself, to establish the existence of pneumoconiosis. §718.202(a)(2). In the instant matter, no biopsy samples were submitted into evidence. Accordingly, I find that Claimant has failed to establish the presence of pneumoconiosis, by the preponderance of the evidence, pursuant to §718.202(a)(2).

C. The Presumptions

If the presumptions described in §§718.304, 718.305 or 718.306 are applicable, it shall be presumed that the miner is or was suffering from pneumoconiosis. §718.202(a)(3). I find that none of the foregoing presumptions are applicable in this matter. Therefore, I find that Claimant has failed to establish the presence of pneumoconiosis pursuant to §718.202(a)(3).

D. Medical Opinion Evidence

A determination of the existence of pneumoconiosis can be made if a physician, exercising sound medical judgment, based upon certain clinical data and medical and work histories, supported by a reasoned medical opinion, finds the miner suffers or suffered from pneumoconiosis, as defined in § 718.201, notwithstanding a negative x-ray. 20 C.F.R. § 718.202(a). Medical reports which are based upon, and supported by, patient histories, a review of symptoms, and a physical examination constitute adequately documented medical opinions as contemplated by the Regulations. *Justice v. Director, OWCP*, 6 B.L.R. 1-1127 (1984). However, where the physician's report, although documented, fails to explain how the documentation supports its conclusions, an administrative law judge may find the report is not a reasoned medical opinion. *Smith v. Eastern Coal Co.*, 6 B.L.R. 1-1130 (1984). A medical opinion shall not be considered sufficiently reasoned if the underlying objective medical data contradicts it. *White v. Director, OWCP*, 6 B.L.R. 1-368 (1983).

In the instant matter, thirteen (13) physicians submitted reports regarding the miner's medical condition. In general, Drs. Previll/Leef, Zaldivar, Fino, Tuteur, Chillag, Dahhan, Castle, and Branscomb found no evidence of pneumoconiosis. While Drs. Rasmussen, Wantz, Stanley, Ducatman, and Bates concluded that the miner had coal workers pneumoconiosis. It is interesting to note that most of the physicians agreed the miner suffered from a totally disabling respiratory condition prior to his death. The disagreement among the experts concerned the cause of the condition.

In general, more weight may be accorded to the conclusions of a treating physician, as he is more likely to be familiar with the miner's condition than a physician who examines him episodically. *Onderko v. Director, OWCP*, 14 B.L.R. 1-2 (1989). Drs. Bates and Stanley were the only treating physicians to render an opinion in this matter and, therefore, their opinions, if well-reasoned and well-documented, may be entitled to greater weight. *McClendon v. Drummond Coal Co.*, 12 B.L.R. 2-108 (11th Cir. 1988).

Setion 718.104(d) codifies the "treating physician rule" and provides the following list of factors in weighing the opinion of the miner's treating physician: (1) nature of the relationship, (2) duration of the relationship, (3) frequency of the treatment, and (4) extent of treatment. Based on the April 9, 1991 medical report of Dr. Stanley (DX 41-25), he had treated the miner for the previous 21 months for his severe respiratory condition. Claimant testified that Dr. Stanley had been the miner's treating physician and that he treated the miner for high blood pressure, breathing difficulties, and cough. Claimant stated that Dr. Stanley prescribed supplemental oxygen and inhalers for the miner. Based on the foregoing, it is clear that Dr. Stanley had been the miner's treating physician for some time and that he treated the miner for his respiratory condition. Therefore, I find that Dr. Stanley has demonstrated he was in a unique position to render an opinion in this matter. However, I find that his opinion is not well-reasoned and well-documented. *Clark v. Karst-Robbins Coal Co.*, 12 B.L.R. 1-149 (1989)(*en banc*). His diagnosis of pneumoconiosis, in his brief medical note in 1991, was based on chest x-ray changes. As noted previously, the vast majority of dually-qualified interpretations were negative for pneumoconiosis. Therefore, I accord the opinion of Dr. Stanley less weight.

Likewise, I accord less weight to the opinion of Dr. Bates on this issue. Dr. Bates was apparently a cardiac surgeon who performed an angioplasty on the miner. It is not known from the record how long he treated the miner, what of the miner's many ailments he had treated, or what treatment he had prescribed for the miner. Based on the foregoing, I find that Dr. Bates has not demonstrated he was in a unique position to render an opinion in this matter. Moreover, I find that his opinion is not well-reasoned and well-documented. *Clark, supra*. He merely stated that the miner had end stage COPD and coal workers' pneumoconiosis that had been documented by his pulmonary team, led by Dr. D'Brot. In fact, the consultation report by Dr. D'Brot referenced the presence of coal worker's pneumoconiosis by history only. Dr. D'Brot reviewed chest x-rays and CT scans, and did not mention findings of pneumoconiosis. (DX 13). A diagnosis of COPD was noted, but neither Dr. Bates nor Dr. D'Brot linked the COPD to the miner's prior coal mine dust exposure. Therefore, for these reasons, I accord the opinion of Dr. Bates less weight on this issue.

I accord greater weight to the opinion of Dr. Rasmussen who authored four (4) reports in this matter. I find that Dr. Rasmussen's final report of June 23, 1998, in particular, is well-documented. In his report, Dr. Rasmussen clearly sets forth the clinical findings and observations upon which he based his diagnosis of coal workers' pneumoconiosis. Moreover, Dr. Rasmussen properly took in to consideration the miner's significant history of approximately 40 years of coal mine employment, as well as his substantial history of heavy cigarette smoking.[11] I also find that the opinion of Dr. Rasmussen is well-reasoned. I find that the underlying documentation contained within his report is adequate to support the conclusion that the miner had coal worker's pneumoconiosis. Dr. Rasmussen noted the disparity in the interpretation of the chest x-rays, but added that the miner showed signs of a progressive impairment of pulmonary function as early as February 1984. He added that there were two risk factors for the respiratory insufficiency: The miner's history of coal mine employment and his significant smoking history. After noting that both risk factors caused similar physiological damage to the lungs, Dr. Rasmussen reasonably concluded that it was impossible to separate the effects of smoking from coal mine dust exposure. For these reasons, I accord more weight to the opinion of Dr. Rasmussen.

I accord less weight to the opinions of Drs. Previl/Leef and Wantz, each of whom examined the miner on one occasion in the 1980s. Their respective reports were based on very limited data in comparison to many of the comprehensive reports submitted post-mortem (*i.e.* reports of Drs. Fino, Zaldivar, Castle, Branscomb, Rasmussen, and Ducatman). *See Stark v. Director, OWCP*, 9 B.L.R. 1-36 (1986). For this reason, I accord the opinions of Drs. Previl/Leef and Wantz less weight.

[11]There are varying smoking histories in the record. Claimant testified the miner smoked two packs of filtered cigarettes per day for more than 20 years, ending in 1993. She did not know whether the miner smoked before they were married. Other reports note two packs per day for 33 to 40 years (DX 24), 1 to 2 packs per day for 27 to 40 years (DX 41-40), and one pack per day from 1969 (DX 41-12). It is clear, based on the foregoing, the miner had a substantial smoking history of at least 1 to 2 packs per day from 1968 (the year the miner was married to Claimant) through 1993, or a total of 25 to 50 pack years.

I accord less weight to the opinions of Drs. Tuteur, Chillag, Branscomb, and Dahhan because their respective opinions are neither well-reasoned, nor well-documented. Each physician concluded the miner had COPD due to smoking. However, each failed to adequately explain how they were able to completely eliminate 40 years of coal mine dust exposure as an aggravating factor, or at least a contributing cause of the miner's COPD, especially when there was evidence in the record that the miner had developed his progressive pulmonary obstruction as early as 1984 (four years before he left the mines). Since COPD is encompassed within the regulatory definition of pneumoconiosis, a doctor's opinion is undermined if it does not give legitimate reasons for ruling out coal dust exposure as a cause or aggravation of that obstructive disease. *Warth v. Southern Ohio Coal Co.*, 60 F.3d 173 (4th Cir. 1995). Moreover, Dr. Dahhan went on to state that the miner's pulmonary disability was due to chronic bronchitis, emphysema, and asthma, which were conditions of the general public and not caused by coal mine dust exposure. However, it is well settled that coal dust exposure can be related to the development asthma and emphysema. *See Robinson v. Director, OWCP*, 3 B.L.R. 1-798.1 (1981). I find that Dr. Dahhan does not adequately explain how he was able to rule out coal dust exposure as a factor in the formation of either disease. For these reasons, I find that the opinions of Drs. Tuteur, Chillag, Branscomb, and Dahhan are not well-reasoned and not well-documented, and as such, should be accorded less weight.

Likewise, I accord less weight to the opinion of Dr. Zaldivar, who opined at his deposition that the miner suffered from lung disease due to asthma and smoking-induced emphysema. As noted above, coal mine dust exposure can be related to the development of asthma and emphysema. *Robinson, supra.* I find that Dr. Zaldivar did not provide an adequate explanation as to how he was able to completely eliminate 40 years of coal mine dust exposure as at least a contributing cause of the miner's pulmonary ailments. For this reason, I find the opinion of Dr. Zaldivar to be less convincing than that of Dr. Rasmussen, and thereby accord his opinion less weight on this issue.

I accord less weight to the opinion of Dr. Ducatman because he opined, without explanation, that the miner suffered from coal workers' pneumoconiosis due to coal mine dust exposure and cigarette smoking, and that he had COPD/chronic bronchitis due to cigarette smoking, coal workers' pneumoconiosis and asbestosis. With regard to his diagnosis of asbestosis, I find that this conclusion is pure supposition and has no basis in fact. Dr. Ducatman admitted at his deposition that he did not personally review the chest x-rays and that there was "some question" as to whether the x-rays were actually consistent with asbestosis. Moreover, I find this diagnosis is at odds with the opinion of every other physician in the record who noted, directly or impliedly, that the miner did not have evidence of asbestosis. Because Dr. Ducatman's report is not well-reasoned, not well-documented, and fundamentally flawed, I accord less weight to his opinion. *See Goss v. Eastern Assoc. Coal Co.*, 7 B.L.R. 1-400 (1984).

I accord less weight to the highly qualified opinion of Dr. Fino on this issue. He concluded that there was no evidence of a coal mine dust-related pulmonary condition, but that the treatment record was one for a lung disease consistent with a smoking-related condition. He stated at his deposition that the pattern and severity of the obstruction was "much more consistent with smoking, and the worsening of his lung function over time was much more consistent with smoking than it was a coal mine related condition." Noting that the pattern and

severity of the obstruction was "more consistent" with smoking does not rule out contribution by coal mine dust exposure. Moreover, I find Dr. Fino's all-or-nothing approach in analyzing the medical evidence unpersuasive. Dr. Fino does not discuss or consider the possibility that both cigarette smoking and coal mine dust exposure could be causing the miner's severe obstructive impairment. For these reasons, I find the opinion of Dr. Rasmussen to be better reasoned and more persuasive than the opinion of Dr. Fino.

I accord less weight to the opinion of Dr. Castle, who is also highly qualified. Dr. Castle acknowledged the miner had two risk factors for the development of respiratory symptoms: Cigarette smoking and coal mine dust exposure. He acknowledged the miner had evidence of asthma and a progressive airways obstruction, which, on some occasions, demonstrated significant reversibility. He concluded the miner did not have the physical findings, the radiographic findings, the physiologic findings, or the arterial blood gas findings to indicate the presence of pneumoconiosis. He added that the reduction of diffusing capacity indicated the development of smoking-induced emphysema. However, at his deposition, Dr. Castle admitted that coal dust exposure could cause COPD, emphysema, and industrial bronchitis. I find that Dr. Castle does not adequately explain how he was able to completely eliminate coal mine dust exposure as an aggravating factor, or at least a contributing factor, to the development of the miner's respiratory ailments (*i.e.* asthma, emphysema). For these reasons, I accord the opinion of Dr. Castle less weight on this issue.

In summary, based on the conclusions of the better reasoned opinions, I find that Claimant has established the existence of pneumoconiosis pursuant to §718.202(a)(4).

E. Weighing the Evidence Together

Pursuant to the holding in *Compton, supra*, I must weigh all of the evidence under §718.202(a) together, in order to make a determination regarding the existence of pneumoconiosis. I found previously that Claimant was not able to establish the existence of pneumoconiosis through x-ray evidence pursuant to §718.202(a)(1). I found there was no biopsy evidence to establish the existence of pneumoconiosis pursuant to §718.202(a)(2), and that the presumptions at §718.202(a)(3) were inapplicable to the facts of the instant matter. However, I found that the conclusions of the better reasoned opinions established the existence of pneumoconiosis pursuant to §718.202(a)(4). After weighing all of the foregoing evidence together, I find that Claimant has established the existence of pneumoconiosis pursuant to §718.202(a).

II. Pneumoconiosis Arose Out of Coal Mine Employment

Pursuant to §718.203(b), if a miner who is suffering or suffered from pneumoconiosis was employed for ten or more years in one or more coal mines, there shall be a rebuttable presumption that the pneumoconiosis arose out of such employment. Employer did not submit any evidence to rebut said presumption. Accordingly, as the miner worked for more than ten years in a coal mine, I find that Claimant has established that the pneumoconiosis arose out of the miner's coal mine employment, pursuant to §718.203.

III. Death Due to Pneumoconiosis

The remaining issue is whether the miner's death was due to pneumoconiosis. Subsection 718.205(c) applies to survivor's claims filed on or after January 1, 1982, and provides that death will be due to pneumoconiosis if any of the following criteria are met:

(1) competent medical evidence established that the miner's death was due to pneumoconiosis; or
(2) pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or the death was caused by complications of pneumoconiosis; or
(3) the presumption of §718.304 [complicated pneumoconiosis] is applicable.

20 C.F.R. § 718.205(c). Pursuant to §718.205(c)(5), pneumoconiosis is a substantially contributing case of a miner's death if it hastens the miner's death.

There is no evidence that pneumoconiosis was the direct cause of the miner's death, therefore Claimant has not proven death due to pneumoconiosis pursuant to §718.205(c)(1). There is no evidence the miner suffered from complicated pneumoconiosis, therefore Claimant has not established death due to pneumoconiosis pursuant to §718.205(c)(3).

There are seven (7) physicians who have rendered an opinion regarding the cause of death. Drs. Rasmussen and Bates found that coal workers' pneumoconiosis was a contributing, or hastening factor in the miner's death. Drs. Ducatman, Fino, Zaldivar, Castle, and Branscomb opined that coal mine dust played no role in hastening the miner's death. No autopsy was performed in this case.

The death certificate listed cardiopulmonary arrest, congestive heart failure, laryngeal cancer, and atrial fibrillation as the immediate causes of death. The certificate was signed by Dr. Stanley, the miner's treating physician. However, a death certificate, in and of itself, is an unreliable report of the miner's condition and it is an error for an administrative law judge to accept conclusions contained in such a certificate, where the record provides no indication that the individual signing the death certificate possessed any relevant qualifications or personal knowledge of the miner with which to assess the cause of death. *Smith v. Camco Mining, Inc.* 13 B.L.R. 1-17 (1989). Although Dr. Stanley had been the miner's treating physician, it is not known what records he reviewed in assessing the miner's cause of death. Moreover, the miner died at home and was not under the direct care of Dr. Stanley at the time of death. Dr. Stanley did not provide any post-mortem report to further explain his conclusions contained within the death certificate. Therefore, based on the foregoing, I accord the diagnoses contained in the death certificate less weight.

I accord greater weight to the highly qualified opinion of Dr. Fino on this issue. At the time of the miner's death, it is clear he was suffering from a variety of medical conditions (*i.e.* respiratory disease, diverticulum rupture and sepsis, laryngeal cancer, congestive heart failure). It is also known that the miner died at home, 40 hours after being released from Mt. Carmel

Medical Center. (DX 24). Dr. Fino noted that without an autopsy, and because the miner died at home, there was no objective evidence of what actually caused the miner's death. He added that because of the multitude of the miner's problems, it would be pure speculation that lung disease, regardless of etiology, was a contributing factor to the miner's death. Dr. Fino reasonably noted that since the miner died at home "how do we know that he did not have a massive stroke, a ruptured abdominal aneurysm, or another heart attack?" I find Dr. Fino's comments in this regard to be very credible and persuasive.

I accord less weight to the opinion of Dr. Rasmussen, who opined without explanation , that COPD was a major contributing factor to the miner's death. Likewise, I accord less weight to the opinion of Dr. Ducatman, who opined death was due to laryngeal cancer that was caused, in part, by asbestosis. As discussed earlier, there is no basis in the record for a diagnosis of asbestosis. I find the opinion of Dr. Ducatman is neither well-reasoned, nor well-documented, and should be accorded less weight.

I find the opinion of Dr. Castle to be equivocal on this issue, and thus, entitled to less weight. He noted that death was due to complications of ruptured diverticulum with sepsis syndrome, and that none of the findings were related to any lung disease. However, at his deposition, when asked whether a disabling pulmonary impairment would compromise a patient's ability to deal with bacteriologic infections, he stated that if a person had developed a primary pneumonia, it would have an impact. He also acknowledged Dr. Rasmussen had mentioned the presence of pneumonia, but added that it would be impossible to know whether the miner had pneumonia around the time of death without an x-ray. Again, this acknowledgement by Dr. Castle supports Dr. Fino's opinion that there was not enough medical information in the record regarding the actual cause of death to make a determination as to whether the miner's severe respiratory condition played any role.

I accord less weight to the opinions of Drs. Zaldivar and Branscomb who, without explanation, concluded death was not hastened, aggravated, or caused by coal mine dust-induced lung disease. Interestingly, Dr. Zaldivar does not opine whether the miner's lung disease, in general, contributed in any way to the miner's death.

Finally, I accord less weight to the opinion of Dr. Bates on this issue. Although he opined black lung hastened the miner's death, Dr. Bates did not specifically identify the cause of death. He alluded to cardiac and pulmonary complications that led to the miner's demise and noted that he "personally" felt that the arrest was a primary pulmonary event. However, Dr. Bates did not point to any medical evidence to support his opinion. While there is no doubt the miner suffered from a severe respiratory condition, it is difficult to know whether the miner's diminished respiratory condition, and complications thereof, actually contributed to the mechanism of death. Again, this leads back to Dr. Fino's conclusion that there was insufficient medical evidence of record regarding the actual cause of death.

Accordingly, based on the foregoing, I find Claimant has failed to establish, by a preponderance of the evidence, that the miner's death was due to pneumoconiosis pursuant §718.205(c).

CONCLUSION

Because Claimant failed to establish all of the requisite elements of entitlement, I find Claimant is not entitled to benefits under the Act.

ATTORNEY'S FEES

The award of attorney's fees, under the Act, is permitted only in cases in which the claimant is found to be entitled to the receipt of benefits. Since benefits are not awarded in this case, the Act prohibits the charging of any fee to the claimant for the representation services rendered to her in pursuit of the claim.

ORDER

The claim of Nelda McBrayer, as surviving spouse of Roy McBrayer, for benefits under the Black Lung Benefits Act is hereby denied.

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ROBERT J. LESNICK
Administrative Law Judge

RJL/LW/dmr

Notice of Appeal Rights: Pursuant to 20 C.F.R. §725.481, any party dissatisfied with this Order may appeal it to the Benefits Review Board within 30 days from the date of this decision, by filing a Notice of Appeals with the Benefits Review Board, P.O. Box 37601, Washington, D.C. 20013-7601. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits. His address is Room N-2117, Frances Perkins Building, 200 Constitution Avenue, N.W., Washington, D.C. 20210.